EXHIBIT 3

DEVELOPMENT OF ASBESTOS-RELATED LUNG DISEASES

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The diseases associated with exposure to asbestos fibers are 1) Asbestosis: Scar tissue in the gas-exchanging regions of the lung; 2) Lung Cancer: Typically (95% of the time) a malignant transformation of an epithelial cell type that lines a conducting airway; 3) Pleural Fibrosis or Plaques: Scar tissue formation in the pleural and sub-pleural connective tissue compartments of the lung; 4) Mesothelioma: Malignant transformation of pleural mesothelial cells.

The lung is comprised of several types of epithelial cells that line the airways and gas exchange areas of the lung. Epithelial cells make up the mucociliary escalator that protects the airways, and the Type I and II alveolar epithelium is the site of initial asbestos deposition and consequent injury at the level of the lung where gas exchange takes place. Mesenchymal cells underlie the epithelial components and produce the connective tissue matrix of the lung. Mesothelial cells line the outside of the pleural membrane (the visceral pleura) as well as the inner lining of the chest wall (the parietal pleura), and these cells line the peritoneal cavity. A variety of inflammatory and phagocytic cells respond to lung injury and mediate components of the developing disease process.

ASBESTOSIS and PLEURAL FIBROSIS:

Inhaled asbestos fibers deposit along all aspects of the respiratory tract. Fibers that land upon a normal mucociliary escalator will be moved to the mouth in a rapid clearance phase. Those fibers that pass through the airways and deposit on the alveolar surfaces can be dealt with in several ways: A) A proportion of the fibers will be cleared onto the mucociliary escalator by the natural movement of surfactant; B) A proportion of the fibers will be picked up by alveolar macrophages which carry the fibers to the escalator for clearance; C) A proportion of the fibers (approximately 20%) will be covered by the Type I alveolar epithelium and drawn into the cytoplasm of these cells for subsequent deposition in the underlying connective tissue (interstitial) compartment of the lung. Some proportion of these interstitial fibers will remain in the lung for the life of the individual while others are cleared slowly over time by interstitial macrophages and lymphatic flow. Shorter fibers are more likely to be cleared from the lung than longer fibers, and short chrysotile asbestos fibers have been found to accumulate in the pleural regions of human lungs. Fibers of all types and all dimensions participate in the pathogenesis of the asbestos-induced diseases.

LUNG CANCER and MESOTHELIOMA:

Inhaled cigarette smoke damages the mucociliary escalator and reduces the clearance capacity of the lung. In chronic smokers there are likely to be areas of squamous metaplasia where the normal pseudostratified ciliated columnar epithelium is replaced by a more simple squamous (flattened) epithelium. There is evidence that a squamous airway epithelium is more phagocytic than the normal columnar type, and this results in the sequestration of increased numbers of inhaled asbestos fibers in the lungs of cigarette smokers.

Asbestos is a complete carcinogen, meaning that it alone is capable of inducing the genetic errors that can lead to cancer. Cigarette smoke contains many such carcinogens. Cancer is the loss of control of cell growth caused by errors in the genes that control cell proliferation. These errors can be induced by direct interaction with carcinogenic minerals like asbestos which causes aneuploidy and other chromosomal abnormalities. Genetic errors also are known to be produced by oxygen radicals that are generated from all forms of asbestos and from components of cigarette smoke. Epidemiology has shown that asbestos-exposed individuals who smoke are more likely to develop a lung cancer than those who only smoke. Since cigarette smoke damages the airway epithelium (as described above), increased numbers of fibers are retained in the airway walls of smokers, and asbestos fibers can bind carcinogens in cigarette smoke, the known carcinogenic effects of both agents can be amplified in a synergistic manner. It takes multiple genetic errors to finally cause an airway cell to develop into a cancerous clone. The chronic exposures characteristic of the asbestos-exposed cigarette smoker is where multiple exposures, day after day, year after year, would be expected to occur. This results in latency periods of 20-40 years as the genetic damage to genes such as k-ras and p53 is repaired, as cells with genetic errors are programmed to die, and as the immune system continues to battle the aberrant cells. In those individuals who are susceptible to developing a cancer, it is obvious that these formidable defense mechanisms have not been sufficient, and at least one cell with a series of genetic errors has escaped to produce the tumor.

Cigarette smoking has no apparent influence on the development of malignant mesothelioma. The only established environmental cause of this cancer in North America is exposure to asbestos. All of the asbestos varieties cause mesothelioma through induction of genetic errors (as described above) in the mesothelial cells that line the pleural or peritoneal cavities. On a fiber per fiber basis, the amphibole asbestos varieties appear to be more potent than chrysotile as causative agents of mesothelioma. Mesothelioma is caused by cumulative exposure and because of this, it is impossible to exclude a particular exposure as contributing to or causing the disease. As with lung cancer, mesothelial tumors typically are derived by clonal expansion of a single transformed cell. Also similar to lung cancer, the latencies before clinical manifestation of mesothelioma are 10 to 50 years or more. Even though mesothelioma is a dose-responsive disease, this tumor has been shown to develop in susceptible individuals with relatively brief exposures, and no safe or threshold level of exposure to asbestos has been determined for mesothelioma. There is no effective treatment for this deadly tumor.